AD 508434

NEUROLOGIC HAZARDS OF DIVING[1, 2]

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Deep sea diving, caisson or tunnel work, and sport diving have one common feature--the high barometric pressure of the environment of the individual. The high pressure introduces several potential hazards (1) to the diver that are related to the gases of the high pressure environment, or to the pressure changes encountered when entering or leaving the high pressure environment. Several of the dangers of diving produce clinical disasters involving the nervous system (2). These may be due to the constituent gases of respiration, contaminants of the same gases or pressure differences within the body cavities and tissues.

When a diver submerges below the surface of the water, the air he breathes must be delivered to him from a compressed source to equalize the pressure of the surrounding water. The compressed air enters the lungs, crosses the alveoli to enter the blood stream and soon reaches the tissues. The amount that enters the tissues is determined by the solubility of the gases in the blood stream, and the diffusion factors of the blood-tissue interface. The absolute quantity of gas that enters the tissues is determined by these factors plus the difference between initial gas pressure and the pressure of the depth of the dive, and the duration of the exposure to the high pressure. The tissues become charged with gas very much like a soda bottle is charged with carbon dioxide. If the pressure driving the gas into the tissues is reduced too rapidly, the tissue gas behaves very much like the gas in the soda bottle when the cap is pulled after shaking: bubbles form in the tissues and the blood stream. The bubbles in the tissue may distort tissue and causes pain, or if they expand rapidly enough

actually tear tissue interfering with their normal function. The
bubbles may also enter the blood stream to obstruct capillaries and
arterioles of various organs. Two definite types of neurologic syn-
dromes develop under these circumstances. One is due to decompression
sickness and the other is due to air embolism.

NEUROLOGIC MANIFESTATIONS OF DECOMPRESSION SICKNESS

Decompression sickness occurs when insufficient time is
allowed for tissue gas to enter the blood stream in solution. The
longer and deeper the dive, the longer the required decompression time.
When the pressure is decreased too rapidly, bubbles form within the
tissues and symptoms are produced by the tissue distortion.

The commonest symptom of decompression sickness is pain.
Those cases that have only pain as the manifestation of disease are
often described as having the "bends". This term appears to have ori-
ginated from the posture of tunnel workers who had compression sickness--
a bent-over posture with hip and knee flexion associated with consider-
able pain in the back and legs. Several other clinical syndromes will
occur and most of them are neurologic in character. If the "bent"
tunnel worker is not adequately treated by recompression he will often
suffer permanent damage to his nervous system. Haymaker has given an
excellent review of the pathologic disturbances found in this condition (3).

The diver presents a somewhat different problem; his neurologic
syndrome is different and the residua of inadequate treatment are usually
different from those of the caisson (tunnel) worker. Divers are re-
stricted in their exposure to high pressures either by a limited gas
supply as with the aqualung, or by the scheduled decompression time

physiologically required, e. g., by the deep sea diver. Those who do
not have pain usually present one of two neurologic syndromes: 1) acute
onset; 2) delayed onset.

The following case illustrates the first or acute onset type.

The patient was decompressing from a 100 foot, 15
minute training dive on air in an indoor training chamber.
At the 7 foot level he complained to his tender over the
telephones that his legs were weak. By the time he reached
the surface of the water he was unconscious. Treatment by
recompression was started immediately and at the 50 foot level
the patient regained consciousness, felt well but weak. At
165 feet, maximum recompression depth, he walked without
difficulty, was disoriented to time and place, but was other-
wise normal. Decompression was started according to the
treatment table schedule, but was complicated by signs of
cerebral edema. This required a readjustment of the decom-
pression schedule. The edema progressed and the patient be-
came hemiplegic and suffered focal convulsions. At the com-
pletion of the recompression treatment that patient was put
in the hospital where he steadily improved over a period of
several weeks.

The diver had a short dive in relatively shallow water and
would not usually be considered to be a candidate for decompression
sickness. His case may actually be an example of air embolism which
will be described later.

The mechanism of this accident is not known. The dive was too shallow to produce classical decompression sickness after the short exposure. The victim had been carefully examined before his training and was considered healthy. A possible alternative to decompression sickness as an explanation for this case was described by Liebow, et al (4), who had the opportunity to perform an autopsy on a case of air embolism, vide infra. A partial bronchial obstruction was found that had gone undetected by history and physical and roentgenologic examinations. Liebow proposed that air became trapped behind the partial obstruction in a single segment of lung, and that on ascent the entrapped air expanded, ruptured the alveolar tissues and gained access to the blood stream with resulting air embolism. Evidence for this type of pulmonary lesion is unlikely to be found by clinical or radiographic methods. Bond (5) proposed that the crucial detection technic would be exposure to high pressure. It is possible that some of the severe neurologic syndromes seen among divers result from such entrapment rather than from the classical mechanisms of decompression sickness. Entrapment is of special interest for those who plan to use hyperbaric oxygen therapy. Patients of uncertain cardio-pulmonary history who may have undetected partial bronchial obstructions will be unsuspected victims of "entrapment". The possibility of air embolism will be high and plans should exist for the correct treatment by recompression.

The second or delayed onset type of neurologic syndrome seen with decompression sickness is less hazardous and somewhat easier to treat. The symptoms appear after surfacing and after a longer delay

than the first type, progress more slowly, and seldom leave residua or
sequelae. The following case is made available to me from the files
of the U. S. Navy Experimental Diving Unit.

An experienced diver surfaced without incident from
his second dive of the day. His decompression lasted 80 min-
utes, and he had been on the surface three-quarters of an hour
when his legs seemed to be numb. One hour later, he had diffi-
culty climbing stairs and in another 30 minutes discovered
that his right leg was very weak. He immediately returned
to the diving site for treatment in the recompression cham-
ber. On examination, he was mentally clear and without speech
difficulties. His right arm was less weak than his leg. The
deep tendon reflexes were more active on the right than on
the left, and the right plantar response was extensor
(Babinski). No sensory deficit was described. He was unable
to urinate. He was recompressed, and at 3 atmospheres pres-
sure had lost all symptoms and signs of deficit. The exam-
ination at this time was normal except for slight weakness
of the right quadriceps muscle. It was assumed that he had
received adequate relief and was therefore treated in
accordance with Table I of the U. S. Navy Treatment Tables.
After surfacing, re-examination was completely normal and he
was symptom-free. Examinations after 24 hours were also
completely normal.

A survey of diving accidents reports we are making is still
unfinished. Unfortunately. it has provided very little information

about specific mechanisms of production of the neurologic syndromes.
The constellation of symptoms referrable to the nervous system is large.
The pattern of their appearance is varied. Seldom do classical neurologic syndromes appear in decompression sickness as they will in major
atery thrombosis or in air embolism. On many instances it is almost
impossible to decide if the disorder lies in the spinal cord, brain
stem, internal capsule or the cerebral hemisphere. A symptom complex
such as blurred vision with red, scintillating scotoma, pain in the
elbow and vertigo is hard to correlate with a single lesion. There
are few neurologic signs or symptoms that have been present in some of
our 500 cases, no characteristic pattern has been found, and yet few
classical clinico-anatomic patterns have been seen.

NEUROLOGIC MANIFESTATIONS OF AIR EMBOLISM

Air embolism among divers is a very real danger and very
serious accident. It usually occurs when ascent is rapid, which in
itself is often an accident, and is not related to the quantity of gas
in the tissues (6). The accepted mechanism for the production of air
embolism is rupture of the alveolar wall by over-expanded gas within
the lungs and an increased transpulmonic pressure (7). The entrapment
mechanism is one possible cause of over-expanded gas. Breath-holding,
coughing, swallowing are others. When the diver has his lungs filled
with compressed gas and ascends more rapidly than this compressed gas,
now rapidly expanding as the pressure decreases, can escape through the
trachea, the alveolar tissues will rupture and gas will enter the blood
stream. Less often, the gas will dissect the peribronchial tissues and

enter the mediastinum. The air embolus enters the arterial stream, passes through the heart and usually enters the cerebral circulation. The syndrome evolved is determined by the location of the embolus and any changes that may occur in the embolus after it first obstructs a vessel. The clinical onset is sudden, occasionally while the subject is still in the water, and always dramatic. Treatment is immediate recompression to 6 atmospheres according to the U. S. Navy Treatment Tables (1).

Among divers the commonest signs and symptoms of air embolism occur almost immediately on surfacing, but one-third occur in ascent in the water. The commonest initial symptom is loss of consciousness, but a dizzy feeling, pain, paralysis, paresthesias, convulsions, chest pain, cough, nausea and headache may occur before loss of consciousness. Some patients have visual symptoms such as scotoma, constriction of the visual fields, loss of vision, or changes in color perception suggesting emboli in the vertebro-basilar system. One patient reported an hemianopsia. Regardless of the mode of onset, if the patient is not unconscious from the beginning, he will usually lose consciousness and frequently will have a convulsion. Hemiplegias are common, and respiratory distress is frequently noted.

The only large collection of clinical data available to us is from the submarine escape training program of the U. S. Navy. Air emboli undoubtedly occur among sports-divers, using aqualungs or other breathing devices, but no reporting system exists to furnish the information.

There is little doubt that the neurologic syndrome is produced by obstruction of parts of the arterial supply to the brain. The obvious

need to reconstitute satisfactory blood flow to the embarrassed brain tissue requires haste in initiating treatment. Fortunately prompt treatment is highly successful, and not only are deaths uncommon when correct treatment is used, but post-treatment morbidity is rare (6). The perfect treatment plan requires recompression within two or three minutes of the clinical onset. The following patient typically exemplifies cases of air embolism.

An experienced submariner was learning to make free ascent escapes. At the training tank he successfully made several free ascents from shallow depths. During the 40 foot ascent, no errors in technic were noted by the instructors in the water. He surfaced uneventfully and swam about 8 feet to the side of the tank. Thirty seconds later he was clinging rigidly to the ladder with the head turned sharply to the left and a glassy stare was observed. He was unable to speak and appeared to be out of contact with his environment. His hands were pried from the ladder, he was taken into the recompression chamber, and rapidly recompressed to 6 atmospheres pressure within 4 minutes of the onset. At 2.8 atmospheres he began to move freely and at 4 atmospheres was considered symptomless. Physical examination revealed no abnormalities. Treatment was executed successfully and the patient recovered completely.

Without apparent warning, the diver experienced sufficient focal brain damage to produce an adversive movement, loss of contact,

and very probably a focal seizure. He had amnesia for the entire
episode. This is the typical clinical picture seen by the diving in-
structors of the world: sudden onset, rapid and successful therapy,
and no sequelae.

A second example of air embolism is presented to illustrate
the clinical variability and to offer some indirect evidence for the
etiologic mechanism.

A 35-year-old man who had completed submarine
escape training several times was undergoing refresher train-
ing as required. He left the 50 foot lock at the escape
training tank without difficulty for a free ascent. During
the ascent, for reasons not stated in the report, he was
taken into the 18 foot lock and when seen there was sputter-
ing and coughing. He was rational and without apparent
neurologic deficit. The lock was drained and he was taken
to the recompression chamber in about 2 minutes. There, his
pulse and skin color were good, but his breathing was ster-
torous. Limpness developed almost immediately in the left
limbs followed by stiffness in the right. Then generalized
flaccidity except for trismus followed and marked nuchal
emphysema was noted. Recompression was immediately started.
The patient was at this time unconscious and failed to respond
to stimuli. At 5.2 atmospheres, he was semiconscious but
failed to recognize those around him, and he did not respond
to verbal commands. At 6 atmospheres, he regained conscious-
ness and shortly thereafter muscle tone returned and breathing

became easier. The patient stated he felt comfortable. The left limbs were described as numb but this complaint disappeared after 20 minutes exposure at 6 atmospheres. The treatment was continued and completed without further event, but for signs of mediastinal emphysema. Post-recompression x-rays of the chest revealed streaking in the pulmonic fields and mediastinum compatible with emphysema and atelectasis.

This patient experienced severe and rapid neurologic embarrassment. Because the neurologic disturbances were so immediately associated with pulmonic symptoms and signs, it could be predicted that they resulted from air embolism of pulmonic origin (8). Their bilaterality suggested either bilateral carotid or vertebro-basilar emboli. The very short duration of the clinical disorder was due to the immediate use of correct therapy. There is very little evidence concerning the allowable safe delay before therapy, but if one assumes that the symptoms were the result of arterial obstruction, then only a few minutes is available before irreversible damage will occur. Therapy now available and established as effective is recompression in a properly manned treatment unit. No other technique appears to remove the emboli as effectively as recompression. Animal experiments reported by the French Naval Laboratories at Toulon suggest that Heparin when quickly injected may be equally useful. The residua of air emboli decreased from about 90% in their unheparinized animals to almost 10% in the heparinized animals. Whether this method can be used in the human is not yet demonstrated. Understanding of the mechanism of the benefit

from Heparin is at best speculative but a decrease in bubble surface
tension and a consequent fragmentation of the bubbles with relief of the
obstruction may be suspected.

MANIFESTATIONS OF INERT GAS NARCOSIS

Behnke, Thomson and Motley (9) demonstrated that the confu-
sion and narcosis experienced by deep sea divers breathing compressed
air was due to the nitrogen in the air. They were able to show that
heavier inert gases (argon) increased the narcosis and lighter gases
(helium) decreased it. According to the Meyer-Overton hypothesis for
the narcotic effect of volatile gases, the oil-water solubility ratios
of the agents are related to their efficacy. The measure ratios, called
the Bunsen coefficients, of the inert gases predict the narcotic effect
in the order observed. These studies have been confirmed frequently.

The clinical manifestations of inert gas narcosis are protein
and variable. Prolonged exposure will produce in the subject an unmis-
takeable clinical state of mental ineffectiveness, euphoria, inappro-
priate behavior, lethargy, hallucinations and unconsciousness.

Susceptability to narcosis is individual, variable from time
to time and from person to person, and rarely noted at pressures of
less than 4 atmospheres when breathing air. Bennett (10) reported that
electroencephalographic features may predict susceptability in certain
cases, but this has not yet been confirmed.

MANIFESTATIONS OF HYPEROXIC POISONING

Exposure to high partial pressures of oxygen produce toxic
effects on various tissues of the body, occasionally manifest by physio-
logic alterations alone and occasionally accompanied by histologic

changes. Paul Bert (1878) described oxygen convulsions (11). Lorrain
Smith described the pulmonary damage after 0.8 atmosphere oxygen expo-
sure of several days (12). The tragedy of high oxygen environments in
incubators for premature infants that "caused" retrolental fibroplasia
is familiar. Viruses grow better when the partial pressure of oxygen
is increased (13). De Almeida described testicular damage in experimental
animals after exposure to high partial pressures of oxygen (14). Steri-
lity may become an occupational hazard among surgeons who use hyperbaric
oxygen chambers.

The type of hyperoxic poisoning seen among divers occurs when
the partial pressure of oxygen exceeds two atmospheres absolute and
takes the form of the oxygen convulsion (15). A diver working at 30
to 35 feet on pure oxygen may experience a convulsion whereas the same
man, sitting quietly in a dry chamber, may tolerate exposures to 60 or
70 feet for periods two to three times as long. It has also been
observed that more divers have convulsions in warm than in cold water.
A small increase in the partial pressure of carbon dioxide in the breathing
medium will also shorten the latency and increase the incidence of con-
vulsions. Exercise will also increase susceptability to oxygen convul-
sions. Oxygen is rarely used to replace compressed air by non-military
divers because of the hazard of oxygen convulsions. Sports divers should
never use oxygen in their equipment, but in the event of their becoming
afflicted with decompression sickness may be given oxygen under increased
pressure during treatment. Unfortunately, even individuals with experience
using oxygen during treatment may still on any particular exposure have
an oxygen convulsion (16). Fortunately, the frequency of such an event-
uality is very small.

The cause of the convulsions, or the mechanism of their production is not known. Several theories exist, but little clarification has occurred. The clinical manifestations will sound quite familiar to clinicians (especially the neurologists) as they are little different from those observed among patients with epilepsy. Many of the patients can tell you that they became hot, felt flushed, had a constriction of their peripheral visual field, developed a sudden headache (generalized), noticed twitching in the facial muscles, especially about the mouth, or developed a generalized paresthesia--a numb feeling, not a tingling. Some had vertigo, while a few experienced nausea, tinnitus, or scintillating scotoma. About 30 per cent of the patients will lose consciousness as the first neurologic manifestation of the oxygen poisoning. Most of the convulsions have been described as tonic-clonic, generalized, motor seizures, indistinguishable from those of grand mal. I have found one or two that are focal, but this is unusual.

If the convulsion occurs in the water the greatest hazard is drowning. This is one of the many safety reasons for diving with a "buddy". Treatment of the oxygen convulsion is straight-forward--stop using oxygen; it is not necessary to decompress. Over 95% of the convulsions were self-limited in our series, and no specific therapy beyond replacing the oxygen with compressed air was given.

Several studies have been conducted on the effect of drugs on the incidence of the oxygen convulsions. Most of the anticonvulsants have increased the threshold, and many agents that increase the partial

pressure of carbon dioxide in the tissues will decrease the threshold. One theory of oxygen toxicity postulates impairment of sulfhydral containing enzyme systems, as may occur after radiation, and therefore several agents used to prevent radiation sickness have been tested and found to offer some protection against oxygen toxicity.

Fortunately, this neurologic hazard of diving can be avoided by the simple expedient of using compressed air for the breathing medium, and using safe diving procedures while in the water.

UNCOMMON SOURCES OF NEUROLOGIC HAZARDS

When a diver is not careful of his source of compressed air, he may find it contaminated with carbon monoxide. This occurs when oil-lubricated compressors of ordinary design are used. Divers' gas supplies may be obtained from special compressors, and these should have their compressed gas checked by routine gas analysis. Carbon monoxide contaminated gas supplies when breathed at depth may result in unconsciousness or death without warning. Treatment is immediate removal of the contaminated gas source and thus resuscitation measures indicated.

Many examples of marine life produce nervous system toxins that can be hazardous to the diver. The sea snake is perhaps the most toxic, but the sea wasp, the Portuguese man-of-war and sea urchins are similarly dangerous. The symptoms often start with intense pain (man-of-war, urchins, sea wasp), followed by paralysis, anesthesia, loss of speech, respiratory embarrassment, convulsions and death. The treatment consists of removing the toxic source, cleaning the wound, occasionally administering antihistamine drugs, and in some instances

antivenom (sea snake). Several fish are venomous. The symptoms are
quite similar, as is the treatment. Prevention is the best therapy.
Prompt recognition of the marine hazard permits the diver to avoid it.
Gloves, exposure swim suits, and shoes will often protect the sports
diver.

SUMMARY

Diving is hazardous. Both the water and the increased pres-
sure present hazards to the nervous system. Some of the neurologic
manifestations of decompression sickness--air embolism, inert gas nar-
cosis, and oxygen toxicity--have been described. A few of the marine
life hazards have been mentioned that are toxic to the nervous system.

Careful diving technic combined with knowledge of the potential
sources of danger should permit safe diving without embarrassment to
the nervous system. Carelessness in diving is fraught with danger.

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